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Pulmonary Function Changes in Children Associated with Particulate Matter Air Pollution from Wood Smoke

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Introduction

Air pollution composed of fine particulate matter may be a risk factor for decreased lung function and increased prevalence of respiratory disease symptoms, especially in young children under 12 years of age. For instance, a two year study of the relationship between pulmonary function changes in third and fourth grade children and air pollutant alerts in Steubenville, Ohio, found a decline in pulmonary function tests associated with increasing 24-hour concentrations of total suspended particulate matter (TSP)[1]. Peak values of TSP ranged from 27 $\mu g/m^3$ to 422 $\mu g/m^3$. The pulmonary function declines were small but persisted for up to two weeks. Similar findings were reported from the Netherlands in a study of children aged 6-11 years before and during an air stagnation episode[2]. Data reported by Ware and associates[3] show increased respiratory symptoms in children aged 6 through 9 years exposed chronically to elevated levels of particulate matter (as well as sulfur dioxide).

The sources of particulate matter in the above studies were mainly industrial and automotive. Another growing source of particulate matter in residential neighborhoods is wood burning stoves and fire place inserts. Many communities, including Seattle, have geographical areas with high concentrations of fine particulate matter from wood burning during the winter heating season. There is wide-spread concern that this source of air pollution may cause or aggravate respiratory illness.

Studies in the US have explored the relationship between wood smoke and health. Honicky and co-workers[4] conducted a survey study of 31 preschool children who lived in homes using wood stoves and 31 children whose homes had no wood stoves. A significant increase in severity of respiratory symptoms was seen in the exposed children. Butterfield et al.[5] also found increased symptoms, especially wheezing, in children living in homes with wood stoves. The use of a wood-burning stove was found to be a risk factor for lower respiratory tract infection in American Indian children[6].

The evidence is not completely consistent, however. Tuthill et al.[7] found no association between wood stove usage and symptoms of respiratory disease. A limitation of prior research is that none of the above studies provided either indoor or outdoor air monitoring data.

Larson et al.[8] measured particulate matter less than 10 micrometers (PM_{10}) emitted by wood burning devices for three heating seasons at a residential site in north Seattle. Due to the topography of the city, some areas along creek drainages have quite high accumulations of PM_{10} on clear nights, whereas nearby areas on ridges have 2-3 times lower concentrations. A questionnaire study conducted in both these areas suggested that

young children aged 1-5 years living in the high wood smoke area had a pattern of more respiratory symptoms and disease than similar children living in the less polluted area[9]. In order to evaluate further possible respiratory effects on children, we conducted repeated spirometric measurements in young children differentially exposed to wood smoke during two heating seasons. Statistical analysis clearly indicated a strong association between light scattering coefficient, a surrogate of PM_{10}, and declines in pulmonary function. This was true for all children, but the association was much stronger in children with asthma. We conclude that exposure to fine particulate matter from wood burning stoves in residential neighborhoods causes decreased pulmonary function in young children. A preliminary report of these data was presented last year[10].

Methods

The study extended over two heating seasons, 1988-89 and 1989-90. The subjects during the first study year were 313 children, including 26 with asthma, from two participating elementary schools drawn from the target area. During the second study year, only 26 children with asthma were studied. Fourteen of these asthmatics had been studied in year one; the remaining 12 children had either graduated to seventh grade or moved from the schools. Consent forms describing the study were sent home with all third through sixth grade children. In the first year, all children who returned a signed consent were studied; in year two, all asthmatics whose parents consented were studied. Table 1 summarizes pertinent characteristics of the children.

Questionnaires were sent to nonparticipants to determine whether they differed as a group from participants. Nonparticipants mainly did not enter the study because they did not receive the form. There were no differences regarding parental smoking, presence of asthma, or household use of a wood heat between the two groups.

Spirometry was measured in each school during morning recess or the lunch hour on four occasions. From these records forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) were calculated. Height was measured at each period, and the child was asked if he or she had been ill during the preceding week. Information on parental smoking, the presence of a wood stove in the home, allergy, or asthma were obtained on the consent form. Baseline lung function measurements prior to the heating season were conducted in September. The lung function measurements were gathered again in the first week of December, and third week of February, and the third week of May. During year two, additional measurements were made in December 1989 and in January 1990.

The spirometers used were three OHIO 822s and one Vitalograph. Even though the Vitalograph spirometer was

computerized, all tracing were hand calculated to be comparable with the manual spirometers. Each child was asked to perform three acceptable tracings. The best FEV_1 and FVC from each record was used. All values were converted to standard body temperature pressure saturated (BTPS) values.

Air monitoring data during the first study year were collected with integrating nephelometers at three sites, one each in the high and low wood smoke areas and one intermediate site. Also filter samples collected using Harvard-EPRI samplers with 2.5 µm inlets. Light scattering coefficient (b_{sp}) has been shown to be highly correlated with PM_{10} at these residential locations ($r^2 = .9$)[8]. For the statistical analysis, the nephelometer record from the high wood smoke site for the day preceding each functional measurement period was used. A random effects model for repeated measures was selected for studying the relationship between b_{sp} and FEV_1 or FVC. The model is based on the method described by Laird and Ware[11] and was carried out by the Restricted Maximum Likelihood Estimation (REML) program obtained from the Harvard School of Public Health. The logarithmic transformation was performed on the light scattering (b_{sp}) data before model fitting in order to alleviate the skewness of the b_{sp} distribution. Since each child acts as his/her own control in the growth curve models, no adjustments for individual characteristics (e.g. height, gender) are necessary.

<u>Results</u>

Air monitoring data from the high wood smoke area show that the average PM_{10} from November, 1988 to March, 1989 in the first study year was 44 µg/m^3. During the second study year, there were frequent windy days and the average PM_{10} was lower, 38 µg/m^3. The average light scattering values during the first and second heating seasons for the day preceding lung function measurements were 2.46 and 0.81×10^{-4} respectively.

Table 1 shows the grade distribution of the 343 children who participated in study. The table also shows the number of children with asthma, allergy, parental smoking, and household use of wood heat.

The estimates of the population slopes for b_{sp} versus FEV_1 and FVC are shown in Table 2. Because of significant interactions between asthmatic status and the slopes, separate results are presented for asthmatic and nonasthmatic children. All slopes are significant with greater significance found for values from children with asthma. The relationship between FEV_1 and FVC and light scattering is significant at the 1% and 5% level respectively. The results for children with no parental smoking, not shown here, were similar to those of all children, indicating that parental smoking was not a confounding factor.

Table 1. Characteristics of the Children

	N	Allergy w/o Asthma	Asthma		Smoker in home	Wood heat in home
			1988-89	1989-90		
Third Grade	109	18	7	5	13	40
Fourth Grade	61	16	6	4	14	20
Fifth Grade	98	10	7	6	12	40
Sixth Grade	67	23	6	7	14	22
Total N	335	67	26	22	53	122

Table 2. Estimates of slopes for FEV_1 and FVC versus light scattering coefficient.

	Asthmatics		Nonasthmatics	
	FEV_1	FVC	FEV_1	FVC
Slope estimate (Liters)	-0.089	-0.097	-0.026	-0.032
Standard error	0.026	0.025	0.011	0.010
Slope/std error (Liters/(10^{-4}/M)	-3.401*	-3.96*	-2.35#	-3.19*

* p = 0.01 level
\# p = 0.05 level

Discussion

This analysis clearly indicates that increases in air pollution are associated with declines in children's pulmonary function. In addition, these effects are far greater for asthmatic children than for nonasthmatic children. There is growing evidence that exposure to constituents of wood smoke affect pulmonary function in young children. The pollutants emitted into the air when wood is burned are products of incomplete combustion. In this regard, wood smoke resembles environmental tobacco smoke, for which numerous studies have shown deleterious effects on respiratory health of children[12]. Both pollutants contain nitrogen oxides, carbon monoxide, benzo(a)pyrene, aldehydes and various polycyclic aromatic hydrocarbons[13]. In the present study, the fine particulate matter portion of wood smoke pollution (b_{sp}), which was found to be highly associated with depressions in pulmonary function over the heating season. However, it is possible, that fine particulate matter is not the only constituent of wood smoke which is a respiratory irritant.

There is one report of considerable acidity in wood smoke particles[14]. A recent study in the Seattle air shed measured hydrogen ion from filters placed in the high wood smoke area of our health effects study[15]. There is evidence that fine particles in the form of sulfuric acid cause decreases in pulmonary function in adolescent asthmatic subjects after a brief 45 minute exposure[16]. Therefore it is biologically plausible that aerosol acidity was responsible for the effect seen in this study.

In studies of associations between air pollutants and respiratory health, it is always possible that some confounding variables are biasing the outcome. Our analysis indicated that removing data from children exposed to parental smoking did not change the analysis.

Another important result of this study, is the increased risk from community pollution attendant to children with asthma. Table 3 shows that the associations between b_{sp} and both FEV_1 and FVC changes are much stronger for children with asthma than for their nonasthmatic peers. Other studies of the effects of community air pollution on respiratory health in children also have suggested increased susceptibility among asthmatics. Dockery and co-workers[17] reported that chronic cough, bronchitis and chest illness were positively associated with fine PM, including fine sulfates, in a group of children studied in the Harvard Six City Study. Although the association between PM and symptoms in asthmatic children was not significantly different from the nonasthmatic children, the authors concluded there was a stronger association. With regard to pulmonary function, Stern and others[18] reported significantly lower FEV_1 and FVC values in children aged 7-12 when comparing two communities with different levels of air pollution. They also saw a significant relationship between air pollution and wheeze, and a significant relationship between use of a gas cooking stove and asthma.

Since the air monitoring period chosen in this study was the evening before the pulmonary function measurements for each child, our results most likely indicate an acute effect of the air pollutant rather than a chronic one.

We conclude that the airborne concentration of fine particulate matter measured by light scattering in a wood burning community is strongly associated with retardation in pulmonary function growth in children exposed during a winter heating season. Further more, the relationship is strongest for children with asthma.

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NOTE TO EDITORS

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